The Guillain-Barré Disease Complex

An Analysis of the Disease with Therapeutic Suggestions and Report of 26 Cases

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• **The Guillain-Barré disease complex may result from a number of causes and have a wide variety of effects. The basic mechanism seems to be an immunizing or allergic reaction to many pathogens or their products, causing edema of the nerve roots in the spine, specifically about the meningeal covering. Resulting pressure on the axon causes nerve damage proportional to the severity and duration of pressure.**

Results in the 26 cases here reported and in other reports indicate that corticosteroids are the treatment of choice, the purpose being to reduce edema as promptly as possible. As might be expected, this therapy is of little value in the post-inflammatory stage of the disease, although prophylactic administration should continue for several months.

Nerve and muscle rehabilitation are the chief aims of later treatment.

AMONG THE imputed causes of the Guillain-Barré disease complex, infectious diseases are most frequently mentioned, especially the viral.[18] Wilson thought in 1918 that he had cultured the causative virus,[3] but his findings have not been confirmed.[17] More recently Bergamasso and Bottiglioni reported culturing a neurotropic virus from patients with this disease, according to von Hagen, who also mentioned enzyme disturbances and toxicity such as heavy metal poisoning among imputed causes.[18] Wider afield are the associations of the disease with serum sickness, postvaccinial sequelae, antibiotic reaction, cervicodorsal arthritis, diabetes mellitus, artificial fever, serum potassium excess in renal failure and malignant growths.[12] In a case observed by the author the features typical of Guillain-Barré disease were manifested immediately after a severe electrical shock.

Some of the foregoing associations must be due to mere coincidence. In others a well-recognized causal association exists (as for example between cervical arthritis and the nerve-root involvement in Guillain-Barré disease) but does not justify fusion of these entities.[8] In the present state of knowledge it would seem best to consider as Guillain-Barré disease only those conditions in which the cause is unknown and omit those like post-diphtheritic polyneuritis and diabetic neuropathy.

The work of Sabin[14] and Haymaker[9] materially clarified the pathology of this disease. The most consistent findings occur in the proximal portion of the peripheral nervous system where the nerve roots fuse. The sequence of events according to Haymaker is as follows:

a. 1-4 days—edema of the nerve roots.

b. By 6 days—disintegration of myelin and swelling of axis cylinder.

c. By 9 days—lymphocytic infiltration along the cylinder.

d. By 11 days—phagocytosis.

e. By 13 days—proliferation of Schwann's cells.

Following this process regeneration may take place primarily in the nerve roots. However, it is felt that involvement may spread centrally from the primary site to involve the neuraxis or distally along the peripheral nerves. Indeed, in the terminal stage some of the spinal roots and peripheral nerves are completely devastated. These findings complement the clinical observation that where motor symptoms predominate the lesions are primarily in the anterior roots, and where paresthesias complicate the picture both the anterior and the posterior roots are involved.

Regarding the central nervous system the histopathologic findings are less conspicuous:

a. Moderate edema of the brain with acute brain cell changes may occur in fulminant cases.

b. Petechial hemorrhages may occur in the gray matter of the spinal cord (33 per cent in Haymaker's series).

c. Perivascular collections of lymphocytes in the white matter and subependymal tissue (20 per cent in Haymaker's series).

d. The spinal and cerebral leptomeninges may show petechial hemorrhages, hypertrophy and/or hyperplasia of fixed tissue cells. Frequently seen is engorgement of the vessels of the leptomeninges about the cord and nerve roots. Proliferative arachnoiditis about the roots is infrequently seen.

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e. Chromatolysis of anterior horn cells (26 per cent in Haymaker's series).

f. Myelin degeneration of the spinal cord and brain stem may occur.

g. Sympathetic ganglia are reported involved by this disease[9,14] with edema and round cell infiltrates.

Cranial nerves, especially the seventh, the ninth and the tenth, may be affected in the same fashion as the peripheral nerves. The Gasserian ganglion has also been involved.

Other findings are listed below. (The order of the list is the order of frequency as reported by Sabin; the numbers at the end of the line show the number of cases in which the various involvements were reported by Haymaker.)

Heart—myocarditis	7
Kidney—lower-nephron nephrosis (questionably present in 5 additional cases)	7
Adrenals—focal necrosis	1
Muscle—degeneration and/or inflammatory change	4
Liver—focal necrosis	2
Spleen—changes are noted that are compatible with infectious mononucleosis	6

In all three of Sabin's cases he reported acute bronchitis and lobular pneumonia. Haymaker reported that 33 out of 55 patients had bronchopneumonia of hypostatic or aspiration types. These findings corroborate the clinical impression that a fatal ending is most often due to respiratory failure.

The disease is preceded in 50 to 60 per cent of the cases by a clinically apparent infectious process; in the others, it has been suggested, infection also occurs but this prodromal phase remains silent or unrecognized. The nervous system then reacts to the organism or its breakdown products in a hypersensitive manner. In effect, the nervous system reacts to an antigen in a relatively specific fashion. This concept is by no means unique; post-exanthematous encephalitides and acute encephalomyelitis are other examples of allergic reactions of the nervous system (experimental allergic encephalomyelitis). Waksman and Adams[19] were able to reproduce in rabbits a Guillain-Barré-like syndrome by injecting into them an antigenic solution containing a portion of the peripheral nerve of the rabbit. They concluded from their studies that the myelin contained a protein or antigen that specifically affected the peripheral nervous system and that acute infective polyneuritis may have an immunologic basis.

Whatever the nature of the pathogenic agent, the response of the nervous system begins with edema, primarily of the nerve roots and specifically about the meningeal covering. The aperture in the dura becomes choked by the expanded myelin and this in turn compresses the axon. This damage is compounded when the meninges become congested and further compress the radicals. Then the sequence of events previously described occurs and the nerve may either undergo degeneration, if the process continues until irreparable damage occurs, or recovery, if the edema subsides. It is not clear whether or not the edema-impingement-phenomenon is the primary damaging factor or whether cellular reactive degeneration goes on along the entire length of the peripheral nerve.

There is no unanimous agreement on the pathogenesis of the hyperalbuminosis. Reitman and Rothschild[12] believe with Hassin that the spinal fluid is primarily absorbed from the perineural spaces. They feel that if these spaces are relatively occluded by edema or inflammatory reaction there may still be an absorption of spinal fluid but not of the larger protein molecules. Boshes[2] considers the protein excess to be "due to the increased permeability of the dilated radicular and spinal meningeal vessels." He also emphasizes that the edematous reaction at the juncture of the anterior and posterior roots causes stagnation of cerebrospinal fluid and that this may play a part in the accumulation of proteins. Haymaker[9] cites several authors who have noted that where proteins from lumbar spinal fluid are increased, the cisternal fluid protein may be normal. Furthermore, Boshes found that in the second or third aspirate tubes the protein was not as plentiful as in the first. This suggests that the hyperalbuminosis is a local phenomenon. It may also explain why in some classical cases of the disease the spinal fluid protein was normal. The author has observed that the protein may continue to increase long after the acute process of the disease has subsided and even while the patient is recovering. This and the other findings point to the importance of stagnation as a cause of the increase in spinal fluid protein.

CASE MATERIAL

The cases to be reported were observed in the Oakland Veterans Administration Hospital between 1949 and 1958. They include all the recognized cases of Guillain-Barré syndrome and were in various stages at time of admission. Most of the patients were studied and treated on the Neurology Service. The author had the opportunity of personally studying the latter five cases in this series.

SALIENT FEATURES OF THE DISEASE

A recognized prodromal disease occurred in 15 of the 26 cases (58 per cent). The preinfection was considered bacterial in nine cases and viral in six cases. The interval of latency from preinfection to paralytic stage varied from 0 to 8 weeks.

The most frequent symptoms at the onset of the paralytic disease were paresthesias and motor weakness. Nine patients, however, complained of pain, especially in the lower extremities.

Extensive motor paralysis is the rule rather than the exception in this disease. All four extremities were involved by motor weakness or paralysis in 19 cases, and atrophy occurred in eight, most often in the small muscles of the hands.

Although this disease is a form of polyneuritis, sensory response was normal in 10 cases. An unusual finding was a transverse myelitis in three patients which gradually cleared as they improved. This indicates that the intramedullary portion of the cord may on occasion be severely but temporarily involved.

The cranial nerves were affected in 10 cases (38 per cent), the ninth and tenth nerves in seven cases, the seventh nerves in six cases and the third nerves in two cases. Singh and Jolly[16] likewise found that the ninth and tenth nerves were the most commonly involved and that only 24 per cent of their patients had seventh-nerve palsy, although many earlier reports emphasized the high proportion of cases with seventh-nerve involvement.[3,18] It is true that since the paralysis may be subtle and bilateral, it is easily missed. Nonetheless overt seventh-nerve palsy probably occurs in fewer than a third of all cases. Guillain pointed out that "there are cases of polyneuritis associated with facial diplegia which do not belong to the syndrome and in which the etiology is different."[7]

The funduscopic observation of papilledema in relation to this disease complex is rare (it was an equivocal finding in one case in the present series). Gilpin et al.[6] reported two cases in 1936; in the patient who had a cerebrospinal fluid examination no elevation of pressure was found. Drew and Magee[4] reviewed nine cases in the literature and reported one of their own, concluding that the causes of papilledema were still obscure; in four of their cases spinal fluid pressure was within normal limits. In a recent study, Feldman[5] considered increased pressure of the cerebrospinal fluid to be the probable primary etiological factor in papilledema. Yet it is noteworthy that Haymaker reported that the abnormality of the cranial nerves is similar to that found in the spinal peripheral nervous system; that is, edema and cellular reaction. Furthermore, cerebrospinal fluid pressure as measured on lumbar puncture is usually within normal limits regardless of the stage of the disease. These observations support the position that the papilledema is actually a true optic neuritis.

Cerebrospinal fluid was examined in all the cases reported here. Cells numbered 0 to 30; in only nine of 58 examinations did the count exceed 10, and in only two was it over 20. The counts were distributed evenly throughout the course of the disease.

Cell type was recorded in only two of the six examinations in the first week of disease, and in both cases the cells were lymphocytes; this type predominated quite definitely in later examinations, a finding in accord with that of Roseman and Aring.[13] Cultures and virus studies at the state laboratory, done in some cases, were fruitless; other investigators have reported similar negative results.[9,16]

TABLE 1.—*Cells and Protein in Cerebrospinal Fluid at Different Time Intervals of the Disease (In Series Here Reported)*

	Days from Onset of Paralytic Stage				
	0-6	7-15	16-30	30-60	>60
No. of determinations ..	6	5	11	11	19
Prevalent cell type.....	Lymph	Lymph	Lymph	Lymph	Lymph
Mean number of cells...	4.5	8	3	4	5.5
Protein (mg. per 100 cc.)	72	116	142	122	101

Cerebrospinal fluid protein content was 22 to 430 mg. per 100 cc.; often it was normal or only slightly increased in the first week of the disease, rose to a plateau between the third and fifth weeks and then slowly declined, though it may remain abnormally high for years, as it did in one case in the series, and may increase on acute exacerbations of the disease as it did in another case. In only one case among those reported here was the protein content not abnormally increased. In this case no spinal fluid specimens were taken between the second and the seventieth day of the disease. Failure to examine the spinal fluid during the height of the disease probably accounts for the failure to detect abnormal increases in otherwise typical cases that have been reported. Recent investigators have emphasized that more frequent testing virtually always discloses the increase,[13,18] as the findings in the present series corroborate (Table 1). In most cases the increase corresponded to a high colloidal gold curve; in seven there was a mid-zone curve and in five a first-zone curve (with syphilis serologically detected in three of these). Apparently the colloidal gold curve may lag somewhat behind the protein rise.

Albuminuria was detected, as by other investigators,[13] in the acute phase of the disease, as would be expected in patients with muscular wasting and negative nitrogen balance. Total serum protein and its fractions may be little affected early in the disease, but the total protein may decline later because of chronic tissue wasting. Gastric analysis and glucose tolerance tests disclosed little abnormality.

Electrocardiographic changes, primarily T-wave flattening, were detected in three patients, one of whom was found to have myocarditis. Few patients were so tested, although myocardial changes have been recognized, clinically and pathologically, as a complication of Guillain-Barré disease.[12]

Electromyographic studies were made in four cases, in all four disclosing evidence of abnormality in the lower motor neurons. Marinacci[11] states that since the disease must destroy 30 per cent of the

lower motor neurons before it becomes clinically apparent, electromyography may be helpful in early diagnosis.

Electroencephalograms were made on four patients. Findings in one were normal, in another borderline, in the other two characteristic of diffuse slow activity.

Guillain-Barré disease may take one of several courses: Progressive rapid paralysis terminating fatally; rapid fulminant onset and gradual recovery; slow onset and slow recovery; remission and recurrence, which seems related to reinfection rather than to an idiopathic process like that of multiple sclerosis. It may be that the nervous system in the recovery phase is hypersensitive to toxic factors and minor insults that normally could be sustained without neural impairment. Most patients can withstand both viral and bacterial infections without exacerbation of the neural disease. The four courses outlined above cover most cases, the most frequent course being one of slow or rapid onset followed by gradual recovery.

In the series here reported, recovery was complete as early as 10 weeks and as late as 100 weeks after onset (average 37 weeks). The length of observation of the patients who did not recover completely varied from 8 to 98 weeks (average 32 weeks). Ambulation—a definite stage of improvement—was achieved in 5 to 57 weeks (average 29 weeks). The effect of steroids in recovery will be discussed under the subject of treatment.

One patient died of metastatic carcinoma of the esophagus confirmed by biopsy; no necropsy was performed. In the other three cases in which the patient died, complete necropsy was carried out and the diagnosis of Guillain-Barré disease was confirmed. All three died of pulmonary complications —bronchopneumonia in two cases and atelectasis in one.

Two cases previously referred to illustrate the unpredictable effect of intercurrent infection in Guillain-Barré disease. In one of them, while the patient was hospitalized there were two severe outbreaks of carbuncles with pronounced systemic reaction and in one instance with pleural effusion, but there was no exacerbation of neural disease. In the other, however, cellulitis of the right leg was accompanied by facial diplegia, dyspnea and progression of paralysis. Triamcinolone produced a prompt and favorable response and was discontinued after a month. Then a mild upper respiratory infection supervened and caused another exacerbation of the neural disease which promptly subsided when triamcinolone was again administered.

In summary, the Guillain-Barré disease complex usually follows an initiating infection by a period of a few days to weeks. It may occur at any age and there is no seasonal predominance. The paralytic stage usually begins with paresthesia or pain followed by hypotonic paralysis without involvement of the pyramidal tract. Constitutional and sphincter disturbances are rare. Cranial nerve involvement, evident in about a third of the cases, indicates a bad prognosis; a worse sign is dyspnea, present in about one-fifth. Laboratory findings are usually not significant except for the characteristic increase in spinal fluid protein while the number of cells in the fluid remains within normal limits. Electromyographic findings reflect the spinal-root process of the disease while the electroencephalogram may indicate the degree of cerebral involvement. Death is usually due to pulmonary complications.

Guillain has been much criticized for his insistence on albuminocytic dissociation as a necessary diagnostic feature, but in the present series this condition was invariably present in the spinal fluid. If it is not found on several appropriately timed examinations before suppressive drug therapy is begun, the diagnosis should be seriously questioned. On the other hand, the dissociation is only a supportive finding, since it is present in many other diseases.

TREATMENT

Among the many modes of therapy for Guillain-Barré which have not proven effective, two are perhaps current enough to be mentioned. Multiple vitamins have been used for years without any known effect on the paralytic process. Dimercaprol (BAL) has been tried because it restores the enzyme metabolic balance of neurons in such conditions as arsenical intoxication; as late as 1953 von Hagen and Baker[18] suggested that administration of thiamine, crude liver extract and dimercaprol was the treatment of choice. Most of the recent studies do not support the value of dimercaprol.[16]

Antibiotics have of course been used against the primary disease, as also specific remedies (as in diphtheria) when available.

The corticotropins and corticoids were at first proposed nearly concurrently by Seltzer[15] and Stillman.[17] Seltzer and co-workers reported a case of progressive disease with cranial nerve involvement in which dramatic response followed administration of corticotropin and the patient fully recovered in four weeks. Stillman and Ganong studied a similar case with most significant features: On administration of 40 to 80 units of corticotropin daily the eosinophil count decreased rapidly and cerebrospinal fluid protein was reduced in nine days from 132 to 82 mg. per 100 cc. There was concomitant clinical improvement. At this point, though, there was another progression of symptoms with cor-

responding increase in eosinophils and spinal fluid protein. On the theory that the adrenal cortex had become refractory to corticotropin, cortisone was administered intramuscularly at 300 mg. per day. Again there was improvement in clinical and laboratory findings, and the cortisone dosage was gradually reduced to discontinuance four weeks later. Seven months after onset of paralysis the patient had made a nearly complete recovery.

Since its inception many successes have been reported for cortisone therapy. Jackson,[10] reviewing 68 reported cases treated with cortisone, found that early response was often dramatic and that 21 patients were completely well within a month, while only two died. Berlacher and Abington[1] reviewed 24 cases treated with cortisone or corticotropin or both: Of the 16 patients treated while the disease was progressive, 11 had improvement in 48 hours, two others within six days; four relapsed. Of the six treated after the disease had reached a plateau, four were improved in four days. There was no improvement in the three treated during convalescence. Although the reviewers observed no real difference between cortisone and corticotropin therapy in the outcome, relapses during or after discontinuance of corticotropin therapy were common; they accordingly recommended administering 300 to 500 mg. of cortisone daily for two to four days and 100 mg. daily for maintenance thereafter for four to six weeks.

TABLE 2.—*Comparison of the Degree of Recovery in Those Treated with Steroids and Those Untreated*

	Died	No. Recovery	Incomplete Recovery	Complete Recovery
Untreated (20 cases)	20%	15%	50%	15%
Treated with ACTH or Cortisone (6 cases)	0	0	33%	67%

In the series here reported, nearly all patients received multiple vitamins and physical therapy. Four received dimercaprol in therapeutic dosage and in only one case was there (questionable) improvement.

Results with steroid therapy are summarized in Tables 2 and 3. The cases mentioned in Table 3 deserve some comment: Good to excellent results were obtained in four of the five cases in which the disease was progressing. In Case 24 the disease was stationary and no improvement was noted. In Case 23 a four-day course of steroid therapy before admission was said to have been without effect and therefore it was not continued. Despite prolonged triamcinolone administration in Cases 24 and 26, studies of blood and electrolytes as well as of bone structure disclosed no physiologic disturbance.

Steroid therapy is in accord with the concept that Guillain-Barré disease results from swelling of the nerve fibers in the spinal roots during the first five days of the acute paralytic disease or of any exacerbation. Whether this edema be of allergic or infectious origin, the steroids should reduce it. The course of the disease thereafter may be largely the result of injury caused by edema—reactive degeneration. Secondary degeneration begins as early as the sixth day after onset[9] and is accompanied by a cellular response. Peripheral nerve studies have been so few in this disease that the limits of the process are not clearly defined, but there are suggestions that the entire nerve is involved.[19] During this reaction the edema is presumably subsiding, and the disease process has reached its maximal stage. Since the nerve damage is due to the degenerative process and the cellular response, it is not logical to expect dramatic effects from corticosteroids at this stage although they may hasten subsidence of edema and thus save a few nerve fibers from further damage.

TABLE 3.—*Results in Those Cases Treated With Corticosteroids*

Case No.	Treatment	Stage of Disease	Results
13	ACTH 30 units intramuscularly every 8 hours for 4 days	Early, progressing	Good results, no progression in disease and decrease in sensory level. Incomplete recovery
17 (second admission)	Cortisone 100 mg. b.i.d. for 8 days, then Cortisone 25 mg. q.i.d. times 2 weeks	Slowly progressive in exacerbation	Good results initially; eventual total recovery
21	Cortisone, 100 mg. b.i.d continued for three months	Slowly progressive	Good results; complete recovery
23	Prednisolone 10 mg. t.i.d. for four days	Early, progressing	No immediate improvement but eventual total recovery
24	Triamcinolone 6 mg. q8h for 1 month	Maximal disease	No change; incomplete recovery
26	Triamcinolone 6 mg. q8h, two treatment periods, the last continued for 3 months	Progressive	Excellent results; complete recovery

Once edema has completely subsided there is no rational value for the steroids in the regenerative phase; however, there is suggestive evidence that small doses may counteract exacerbations. In Case 26 there was exacerbation on withdrawal of cortisone after successful use in the acute phase, even though the patient was still receiving an antihistamine (diphenhydramine hydrochloride); cortisone therapy was resumed and remission was complete during the subsequent three months of maintenance therapy. In Cases 21, 24 and 26 (Table 3) there were no relapses during long periods of cortisone therapy. Since cortisone does not always bring about remission, it is important to prevent relapse.[10]

In most cases, then, cortisone is the drug of choice. In acute fulminating paralysis when medullary involvement is preeminent, corticotropin (ACTH) should be given intravenously; cortisone may be substituted by the third day if there is good clinical response. The newer steroids—prednisone, prednisolone and triamcinolone—are preferable because they do have fewer untoward side effects. In cases with acute downhill course despite cortisone therapy, a trial of ACTH may be warranted.

Although drug therapy has been stressed in this presentation, physical therapy and other measures are equally important. Following is a brief outline of the treatment at Oakland Veterans Administration Hospital:

A. Immediate measures in the acute phase
   1. Diagnosis by
      a. Neurological examination
      b. Cerebrospinal fluid examination
      c. Indicated laboratory procedures.
   2. Assessment of vital capacity and respiratory function (apply a respirator if any question of respiratory embarrassment exists).
   3. Absolute bedrest.
   4. Positioning in bed with assistive devices, orthopedic mattress with alternating pressure pad, or frequent turnings, and early use of splints and/or bedboard.
   5. Gentle passive exercise of involved muscle groups through a full range of motion twice a day.
   6. Steroid therapy if a question of progressive disease exists.
   7. High caloric, high protein diet.
   8. Salicylates and/or codeine substitutes for pain (barbiturates and narcotics are not necessary).
   9. Prompt treatment of all complications, the most frequent:
      a. Respiratory infections
      b. Myocarditis
      c. Urinary tract infections
      d. Fecal impactions.

B. Steps after acute process:
   1. Manual muscle test and measurements.
   2. Galvanic stimulation to muscles considered moderately weak.
   3. Daily Hubbard tank bath with assistive underwater exercises.
   4. Blow bottles to improve vital capacity if indicated.
   5. Except for above measures, continued bedrest for a total of two months.

C. Rehabilitation after two months:
   1. Physical therapy ambulatory classes; splinting for weak muscle groups.
   2. Continued electrical stimulation to weak muscles.
   3. Supervised exercise in swimming pool.
   4. Self-care evaluation and occupational therapy.

Bedrest in the first two months is intended to allow the earliest maximal regeneration of neural tissue. It is felt that if damaged axons or nerve cells are stimulated, their recovery phase is lengthened. It should be kept in mind that there may be mild cases with rapid recovery which will not require a full two months of bedrest.

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A Simple Way to Drain a Subungual Hematoma

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THE RELIEVING OF PRESSURE in a subungual hematoma nearly always evokes expressions of gratitude from the patient. While the method described below probably is used by many physicians, it seems worthwhile reporting for those who are not acquainted with it.

The instruments needed are a paper clip with one prong bent outward at a 90 degree angle, a Kelly forcep and an alcohol lamp. After the nail plate has been prepared with alcohol or other suitable antiseptic, the paper clip is grasped with a Kelly forcep and the extended prong is heated in the flame of the spirit lamp until it becomes red. With the patient's finger held steady to prevent jerking of the hand during treatment, the heated tip is pressed lightly against the nail plate over the hematoma. Usually it penetrates the nail quite easily and the hematoma drains, with decided relief of pain, as soon as it is withdrawn. Since the heat is readily dissipated as the tip of the clip burns through the nail plate and enters the encapsulated blood, there is little danger of causing a thermal burn of the nail bed.

This method is less painful than drilling of the nail plate, the procedure takes only a few seconds as against five to ten minutes for drilling, and the temperature of the probe provides comparative sterility.

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